HUMAN TOXOPLASMOSIS

Pages with reference to book, From 224 To 226

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The intracellular protozoan parasite, toxoplasma gondii Causes infection in man as well as in animals. The primary hosts which harbour the intestinal, sexual stage are cats[1,2]. Transmission to humans happens mainly by eating raw or undercooked contaminated meat[3,4], raw cows milk and birds eggs or by swallowing oocysts discharged in faeces of infected cats, by close contact with contaminated material, especially cat litter[5,6] or by inoculation of trophozoites through the skin or by inhalation. Transmission from a mother infected during pregnancy to the foetus causes congenital toxoplasmosis[7,8]. Toxoplasmosis is a common infection in humans. In adolescence and adulthood, most infections are subclinical or run a very mild clinical course[9]. Acquired toxoplasmosis is most often associatedwith lymphadenopathy but other symptoms such as fever, atypical lymphocytosis and myalgia may also occur. Chorioretinitis is a well known manifestation of acquired infcction, but may also be a late sequelae of the congenital infection[10]. Toxoplasma encephalitis is also common among AIDS. patients[11]. The most dreaded clinical consequence of toxoplasmosis is intrauterine infection resulting in a congenitally infected neonate. Congenital infection leads to still birth, chorioretinitis, intra-cerebral calcifications, psychomotor disturbances, hydrocephaly and microcephaly. Toxoplasmosis is a systemic infection, always accompanied by the production of serum antibodies, at high titre. After the acute stage antibodies persist at low titre, usually throughout life. The number of seropositive persons in a population therefore, increases with age. Although antibodies for toxoplasma gondii have been found in the sera of humans and animals throughout the world, the proportions of subjects with positive reactions varies considerably by geographic area, age and test method used[1,12]. Antibodies to toxoplasma gondii generally are present more often in adults than in children except in those areas where many infections are acquired in childhood. Antibodies are equally frequent in males and females[19,12,13]. Studies have shown considerable variations in the prevalence of toxoplasma antibodies in different age groups, being more prevalent in childhood in some countries[13,14] and in elderly groups in other countries. In Cleveland[15], 35% of adults between 31 and 4Oyears of age were sero positive and in Syracuse[1], 41% in contrast, about 80% of similarly aged adults in Panama[15]. The difference in antibody prevalence of 10 year old children was 5% in Cleveland and Syracuse and 49% in Panama. High early sero-conversion rates also have been reported from Colombia, Guadeloupe, San Salvador, Costa Rica and other central American countries[15-17]. The sero-conversion in childhood differs from that in adult life, apparently because of the environment or changing habits[15]. Antibody prevalence rose from 25% at 5 years to 50% at 10 years of age and increased gradually reaching 90% by 60 years, in Panama[15]. However, in San Salvador[17], 80% was reached at 35 years, in Costa Rica[18] and Columbia[15], prevalence rates peaked near 60% at 25 years, but in Guadeloupe[16], had already peaked by 10 years. The most common antibody titres in the 20 to 25 year old populations are different in different countries. It is shown that higher titres are related to relative frequency of primary infection in the younger group and to high re-infection rates[15]. Whatever the mechanism, it appears that toxoplasma antibodies usually are acquired from childhood to age 30 to 40 years, after which relatively few such events seem to occur[1]. In pregnant women the prevalence of toxoplasma antibodies was reported 54% in Venezuela[19], 60% in Costa Rica[20], 34% in Mexico City[21], 60% in Tabasco, Mexico[22], 94% in Los Angeles25 and 46% in northern parts of Pakistan[24]. Congenital infection accounts for only a small proportion of toxoplasmosis in htimans in Britain[7] as elsewhere, although differences in the

rates of infection occur and it has suggested that congenital infection in the high lands of Scotland is commoner than in parts of England[7,25,26]. The dissemination of toxoplasmosis in the world does not seem to be confined to specific areas as it has been found where it has been sought, although the frequency is more in the humid tropical regions. The frequency of infection is greatest along the coast of south-eastern Maxico. This may be due to the preference of the parasite for warm zones for reproduction. The frequency of infection by toxoplasma gondii is measured by seroepidemiological studies, in people of different social class, feeding habits and physical environment. The rather unspecific clinical manifestations of the illness make toxoplasmosis including "its congenital form', difficult to diagnose and obviously had to prevent. It has been reported that prevalence of seropositivity among Eskimos is zero; among Brazilians 72%[27]. In countries like the U.S.A. [9], the number who are seropositive vary between 20-70%, in Argentina 62%[28], in Tahiti 68%[29], in city of Paris 84%[30] and in the city of Escuintla, Guatemala 94-100%[31]. These figures are of general population. In tropical countries like Somalia[13] the overall prevalence of antibodies was 56% in the village and 40% in Mogadishu, in Nairobi, kenya[14] the prevalence was 35% in pre- school and 60% in early school age children and in Kuwait 95.5%[32]. The prevalence of toxoplasma gondii antibodies in the Chinese population of Hong Kong[33] was distinctly low, 9.8% which can probably be attributed to the absence of cats in most domestic house-holds and the preference of the people to eat well cooked meat. Studies have been reported from Pakistan concerning prevalence of toxoplasma antibodies in different population groups[24,34-38]. It is obvious that in humid tropical areas the percentage sero positive are more because in hot humid climate, cysts survive longer than in hot arid zones where they dry out[13,15,39] The effective control of infection depends on the accurate knowledge of the transmission. It may be acquired congenitally or by consuming raw and under cooked meat containing cysts or from contact with cats and other animals. Since the early studies in U.S.A. [1,15], the life cycle of toxoplasma appears to have been clarified and transmission by cat-faeces contaminated soil has been postulated. Another element that must be taken into account, are the cities of countries with certain feeding habits. In Europe and the U.S.A toxoplasmosis is acquired mainly by ingestion of under-cooked infected meat particularly from swine, sheep and lamb[9,15,22,26] while in other countries like Latin America, China[37], Somalia[13,39], Kenya[14] and Pakistan[34-38] the living conditions are different. The meat is generally eaten well cooked and pork not at all in Muslim countries, toxoplasmais transmitted via cat faeces. The soil is heavily contaminated by cat faeces, the humid climate favours long survival of oocysts and the subjects come in close contact with the soil. These circumstances greatly increase the risk of human infection by oocysts. Such a route of infection might explain why antibodies are acquired early in life and the infection rate was highest during childhood in some countries. It has been reported[15] that age specific incidence rates were 6.3% to 9.8% per year between 1 and 10 and 11% to 15% per year between 11 and 35 years; thereafter they declined per year probably due to lesser contact with soil contaminated by cats. Transmission may occur through insect vectors like cockroaches and flies[40]. It is important to consider other means of transmission by saliva as a factor in reinfection within family groups[1,41]. Consumption of raw goat's milk can also transfer the infection[42]. Congenital infection has also been reported from different parts of the world[7,8,22-24,30,31]. Hygienic conditions, socioeconomic structure, food and environment can collectively have a notable influence on the diffusion of toxoplasma. The diagnosis of toxoplasmosis can be made by serology, histology of infected tissues or isolation of the pathogen. From a practical clinical point of view, serologic tests are the most readily available. The serological methods commonly used are Sabin-Feldman dye test, the indirect immunofluorescent test and the passive hemagglutination test. The immunoenzymatic assay ELISA IgG, IgM has proved to be a valuable tool in detecting antibodies to toxoplasma antigens. Both IgG responses are produced in infected individuals to toxoplasma antigens. Detection of IgM antibodies establishes the diagnosis of

recently acquired or reactivated infection but these antibodies soon disappear or decrease to very low levels followed by the appearance of IgG which stays longer. Recognition of IgG antibodies, does not help in establishing the diagnosis of toxoplasmosis, since chronic asymptomatic infection can also be associated with high antibody titre of IgG but its detection in serum is of great epidemiological significance, since it will indicate previous contact of the individual with the organisms.

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